Renal care in critical cardiac patient

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Case History

- R.A.M., 66 y old IHD lady, was admitted to ICU of ICC at 7th April 2014.
- Complaint: dyspnea grade 4, orthopnea, generalized swelling, oliguria.
- HPI: frequent hospital admission 5 times in the last 6 mo. Last one was 10 days ago when her cardiologist advised her to be admitted because of failure to parenteral diuretics, raising creatinine from 1.6 to 3.1mg/dl and aggravation of dyspnea.

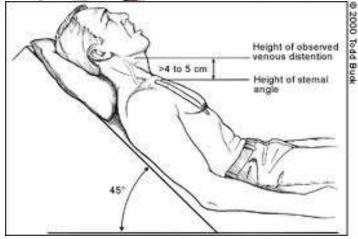
Case History

- Past history:
- ✓ Hypertension since 20 years (no tight control).
- ✓ Gouty on conservative therapy since 16 years.
- ✓ Dyslipidemic since 10 years on statins.
- ✓ IHD with open heart surgery since 7 years.
- ✓ AF on oral anticoagulants since 7 years.
- ✓ Renal impairment since 5 years (cr= 1.6).

Case History

- Drug history:
- > ACE inhibitor and alpha methyl dopa.
- >Anticoagulant.
- > Antiarrhythmic.
- >Statin.
- ➤ Allopurinol and colchicine.
- > Loop diuretic with other class diuretics.





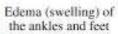














- BP= 160-70 mmHg.
- Pulse= 70 AF.
- Temp= 36.5°C axillary.
- RR=16 /min.

- Chest: wide spread rhonchi, basal to mid-zonal fine crepitations.
- Heart: scar of cardiac surgery, AF, galloping.
- Abdomen: distension, scar of hernial repair, ascites, Peau d'orange, congested hepatomegaly.
- LL: edema, scar of venous graft.
- Neurological: anxious.
- Musculoskeletal examination: OA of both knee, obesity (BMI =31).

- UOP= 20 ml/h.
- Concentrated urine

Investigations

- Creatinine= 1.6- 3.1.
- B urea=170.
- S uric acid= 7.7.
- K= 5.5
- S albumin= 3.2.
- Hb%= 10.5 NN.
- Urine: albumin +, RBCS=15.
- INR= 3.2.

Radiology

- Abdominal US: fatty liver, normal kidneys, ascites.
- Echo: diastolic dysfunction, MR, EF=30%.
- Chest x ray



Renal problems in this lady

- AKI on top of CKD.
- What is volume status management? (hypo or hyper).
- What is ideal RRT if needed?

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AKI on top of CKD

 CKD based on GRF estimation(e GFR by CKD EPI = 33.2ml/min)

| | | | Persistent albuminuria categories Description and range | | | |
|--|-----|-------------------------------------|--|-------------------------|-----------------------------|--------------------------|
| Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012 | | | A1 | A2 | A3 | |
| | | | Normal to mildly increased | Moderately increased | Severely increased | |
| | | | | <30 mg/g =3 mg/mmoi | 30-300 mg/g 3-30 mg/mmol | >300 mg/g >30 mg/mmol |
| GFR categories (ml/min/ 1.73 m²) Description and range | G1 | Normal or high | 290 | | | |
| | G2 | Mildly decreased | 60-89 | | | |
| | G3a | Mildly to moderately decreased. | 45-59 | | | |
| | G3b | Moderately to severely decreased | 30-44 | | | |
| | G4 | Severely decreased | 15-29 | | | |
| | G5 | Kidney failure | <15 | | | |

AKI on top of CKD

AKI based on creatinine and GRF deterioration

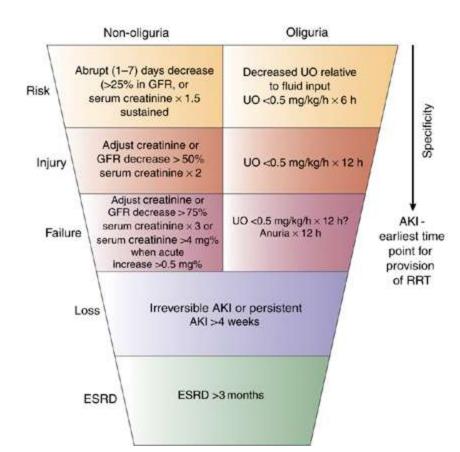
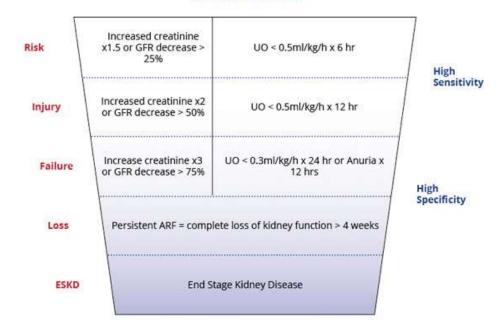


Figure: Rifle Criteria for Diagnosis of AKI

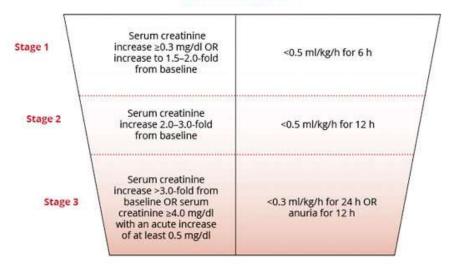
AKI on top of CKD

 AKI based on creatinine and GRF deterioration

RIFLE Criteria



AKIN Criteria



AKI in a cardiac patient Cardiorenal syndrome

 Cardio-renal syndrome means declining renal function in the setting of advanced CHF.

Types of CRS

| | Chronic | Acute |
|-------------|-------------|--------------|
| Cardiorenal | | |
| | Type II CRS | Type I CRS |
| Renocardiac | | |
| | Type IV CRS | Type III CRS |

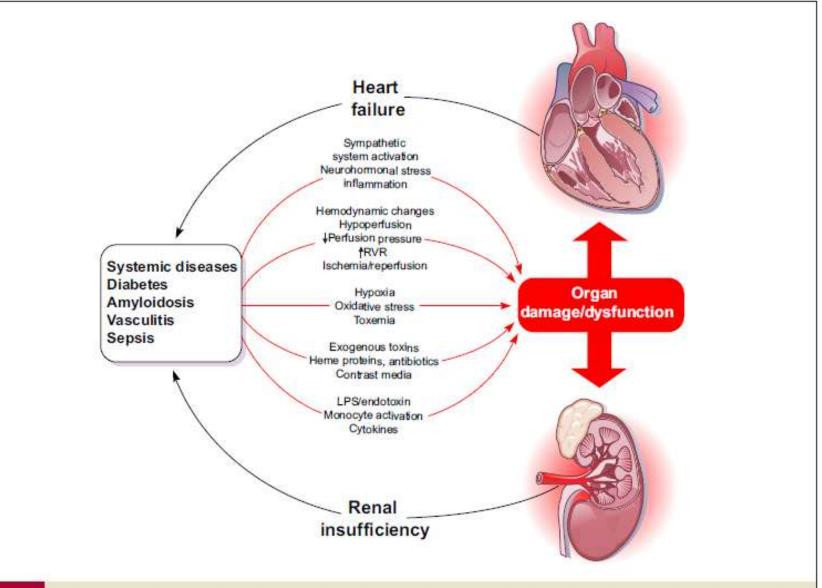


Figure 5 CRS Type 5

Pathophysiological Interactions between heart and kidney in type 5 cardiorenal syndrome (CRS) or *secondary CRS" (systemic condition, e.g., diabetes mellitus, sepsis, causing both cardiac and renal dysfunction). LPS = lipopolysaccharide (endotoxin); RVR = renal vascular resistance. Figure Illustration by Rob Flewell.

Table 1. CRS

General definition

Pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other

CRS type I (acute CRS)

Abrupt worsening of cardiac function leading to AKI

CRS type II (chronic CRS)

Chronic abnormalities in cardiac function causing progressive and permanent chronic kidney disease

CRS type III (acute renocardiac syndrome)

Abrupt worsening of renal function causing acute cardiac disorders

CRS type IV (chronic renocardiac syndrome)

Chronic kidney disease contributing to decreased cardiac function, cardiac hypertrophy, and/or increased risk of adverse cardiovascular events

CRS type V (secondary CRS)

Systemic condition (eg, diabetes mellitus, sepsis) causing both cardiac and renal dysfunction

CRS-Type 1

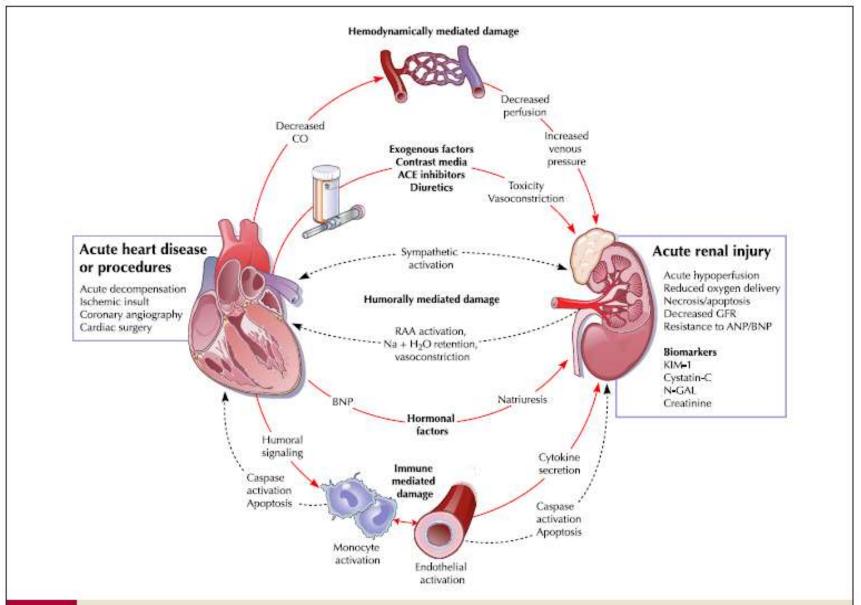


Figure 1 CRS Type 1

Pathophysiological interactions between heart and kidney in type 1 cardiorenal syndrome (CRS) or "acute CRS" (abrupt worsening of cardiac function, e.g., acute cardiogenic shock or acute decompensation of chronic heart failure) leading to kidney injury. ACE = angiotensin-converting enzyme; ANP = atrial natriuretic peptide; BNP = B-type natriuretic peptide; CO = cardiac output; GFR = giomerular filtration rate; KIM = kidney injury molecule; N-GAL = neutrophil gelatinase-associated lipocalin; RAA = renin angiotensin aldosterone. Figure illustration by Rob Flewell.

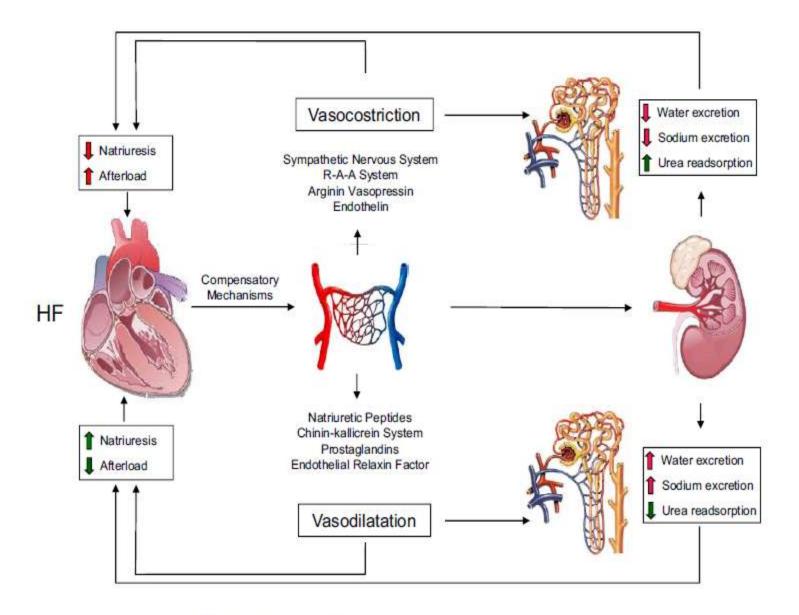


Figure 2. Hemodynamic mechanisms activated in CRS type 1.

Renal problems in this lady

- AKI on top of CKD.
- What is volume status management? (hypo or hyper).
- What is ideal RRT if needed?

Table 2. Overhydration and Congestion: Management With the 5 Bs

Balance of fluids

Blood pressure

Biomarkers

Bioimpedance

Blood volume





Fluid management: the 5 "B"

- Balance
- Biomarkers
- > BIVA
- Blood Volume
- Blood Pressure





BALANCE

Fluid Balance

Daily fluid input:

1.5-2.0 L maintenance

1.5-2.5 L medications

0.8-1.5 L nutrition

0.5-1.5 L boluses



Daily fluid output:

1.0-2.0 L Urine

1.0-2.0 L Insensible losses

1.0-3.0 L Dialysis/ UF

0.5-1.5 L Other

Body Composition

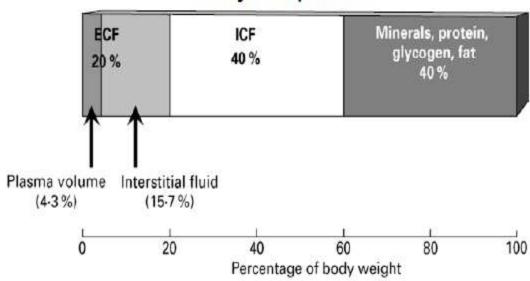


Figure 3. Compensatory mechanisms in HF.



A continuous compromise How to handle a delicate balance?



Daily fluid input:

1.5-2.0 L

maintenance

1.5-2.5 L

medications

0.8-1.5 L nutrition

0.5-1.5 L boluses



Daily fluid output:

1.0-2.0 L Urine

1.0-2.0 L Insensible

losses

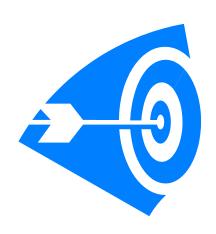
1.0-3.0 L Dialysis/

UF

0.5-1.5 L Other

- In what clinical circumstances? AKI, CRS, SEPSIS, CKD, Dialysis
- How much fluid to give, How fast, What type of fluid? Physiological targets?
- How much fluid to remove, How fast, What modality? (Diuretics VS Dialysis)

Optimal fluid status is our target



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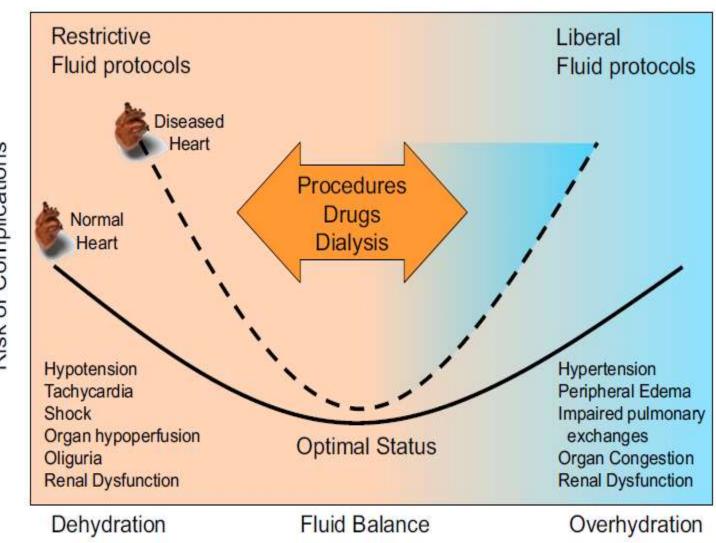


Figure 4. Components of fluid balance calculation and fluid distribution in the body.

Risk of Complications





How should we manage fluids in patients who develop AKI?

Infusions

Diuretics

Extracorporeal ultrafiltration

Blood Volume and hemodynamics

Fluid balance prescription

Fluid exchange prescription

Fluid balance errors





Why are fluids given?

Daily fluid input:

```
1.6 ± 0.2 L maintenance fluids
```

1.8 ± 0.4 L medications

 $0.8 \pm 0.3 L$ nutrition

0.5 ± 1.2 L fluid boluses





Diuretics

- Diuretics can be given to test renal responsiveness after adequate fluid loading
- Diuretics should be discontinued or at least modulated and tailored to each patient if there is no response to avoid side effects.
- There is no evidence that diuretics reduce morbidity or mortality or improve renal outcome
- If urine production is restored, this greatly facilitates fluid management.



The monthly thought of the Editor

Early goal directed therapy and early goal ultrafiltration therapy for critically ill patients with acute kidney injury

Very few people in the world of intensive care medicine have not heard about the Emanuel Rivers Study describing the fluid management approach defined as early goal directed therapy. The study basically concludes that a thorough fluid resuscitation policy guided by hemodynamic monitoring and central venous oxygen saturation in the first six hours of hospital admission is a procedure that improves survival in patients with severe sepsis and septic shock. Early goal directed therapy includes crystalloids or colloids based on CVP measurement vascactive agents based on mean arterial

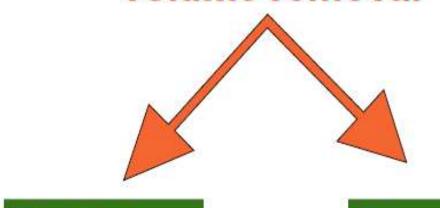
amounts of fluid for resuscitation purposes.

All these observations have pointed out one important aspect of the patients with sepsis and septic shock: the fluid balance is remarkably positive in the first hours of ICU admission. But positive fluid balance has its price: first of all, fluid given in the presence of damaged endothelium and loss of plasma oncotic power makes a real float of the interstitium with swelling of tissues and cells; furthermore, cardiac contractility might be impaired and the fluid overload further contributes to myocardial dysfunction due to hypervolemia. Third, these patients are often olimpic



Treatments for extracorporeal volume removal





Frequency

Ultrafiltration

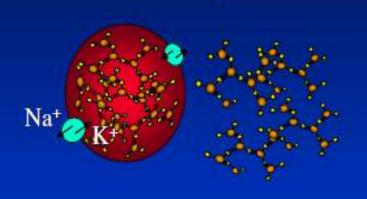
Technique

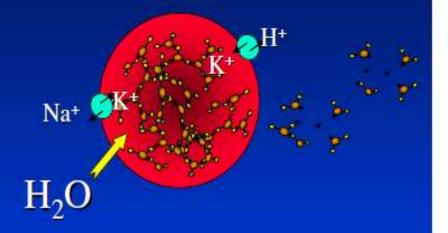
- Hemofiltration
- Hemodialiysis
- Hemodiafiltration

- Isolated
- Intermittent
- Daily
- Continuous

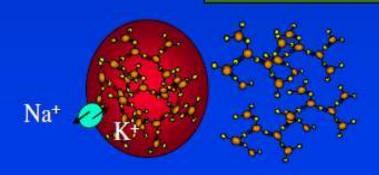
Electrolyte disorders might be worsened by aggressive hemodialysis while they can be progressively corrected by CRRT

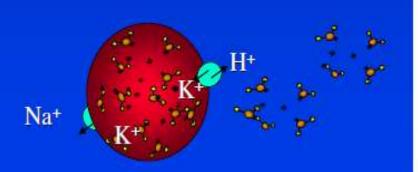
INTERMITTENT HEMODIALYSIS





CONTINUOUS HEMOFILTRATION





Aquapheresis Therapy



Therapy to safely achieve euvolemia (dry weight)

Uses a simplified form of ultrafiltration

- Quick and easy device setup: less than 10 mins - Low blood flow: 20-40 mL/min

Low blood volume: 33 mL

Precise fluid removal rates: 10-500 mL/hour

Inpatient or outpatient settings

. ICU, CCU, MICU, telemetry, stepdown, observation, ED, outpatient clinics

Peripheral or central venous access

· Flexible access sites and catheters

· Diverse physician prescription

 No clinically significant impact on electrolyte balance, blood pressure or heart rate

Think of it as a "mechanical diuretic"...

- Ultrafiltration has been available for decades with CVVH devices
- How often are non-renal, fluid overloaded heart failure patients currently treated with CVVH just to remove excess volume?

Why?

| | Aquapheresis | CVVH |
|------------------------|-----------------------|-------------------|
| Patient | Fluid overload | Renal |
| Treatment Venue | Inpatient/Outpatient | ICU |
| Blood Withdrawal Rates | 10 – 40 mL/min. | 100 – 300 mL/min. |
| Extracorporeal Volumes | 33 mL | 100 – 300 mL |
| Venous Access | Peripheral or Central | Central |





Balance Summary

- Patient's balance is crucial
- Use fluid with great caution
- Consider kidney function
- Use diuretics with individualized modality
- You might be required to remove fluids by Ultrafiltration or other CRRT techniques
- Treatment with extracorporeal techniques should be timely instituted and accurately performed (error-free).
- Techniques are not all the same





Biomarkers

Table 1

Protein Biomarkers for the Early Detection of Acute Kidney Injury

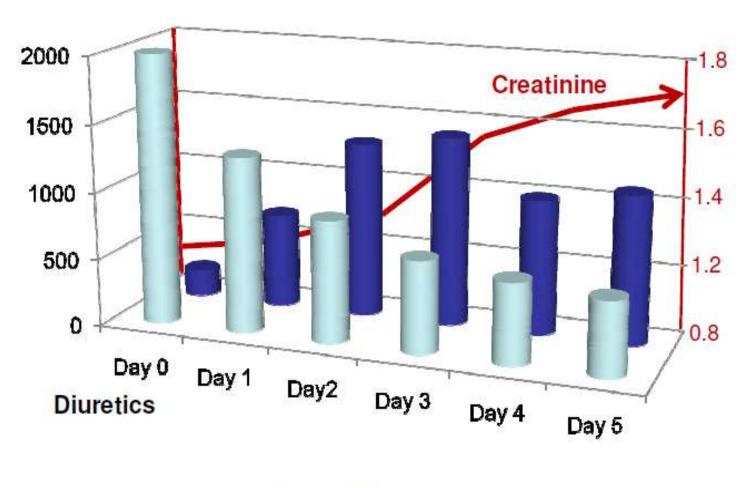
| Biomarker | Associated Injury | |
|-------------------------------|---------------------------------------|--|
| Cystatin C | Proximal tubule injury | |
| KIM-1 | Ischemia and nephrotoxins | |
| NGAL (lipocalin) | Ischemia and nephrotoxins | |
| NHE3 | Ischemia, pre-renal, post-renal AKI | |
| Cytokines (IL-6, IL-8, IL-18) | Toxic, delayed graft function | |
| Actin-actin depolymerizing F | Ischemia and delayed graft function | |
| α-GST | Proximal T injury, acute rejection | |
| π -GST | Distal tubule injury, acute rejection | |
| L-FABP | Ischemia and nephrotoxins | |
| Netrin-1 | Ischemia and nephrotoxins, sepsis | |
| Keratin-derived chemokine | Ischemia and delayed graft function | |

GST = glutathione S-transferase; IL = interleukin; KIM = kidney injury molecule; L-FABP = L-type fatty acid binding protein; NGAL = neutrophil gelatinase-associated lipocalin; NHE = sodium-hydrogen exchanger.





CRS and Diuretics

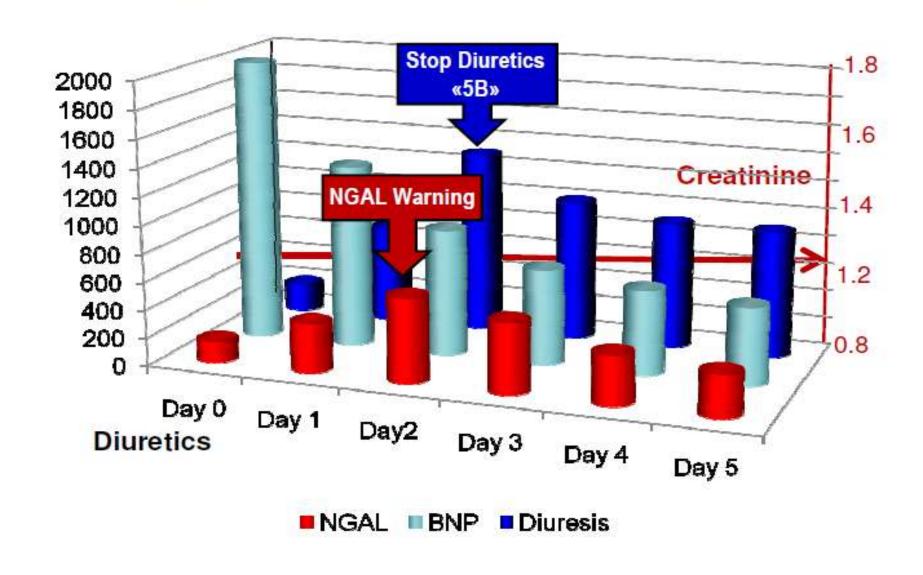


■BNP ■Diuresis



HF, Diuretics and NGAL

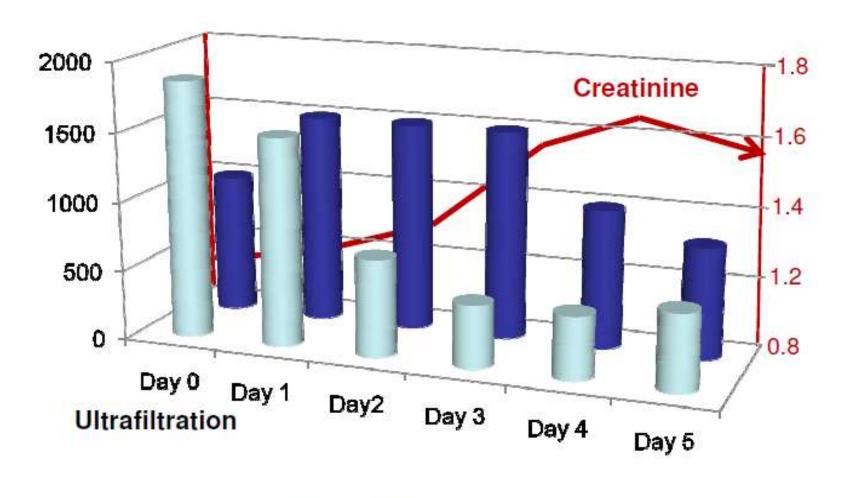






CRS and Ultrafiltration

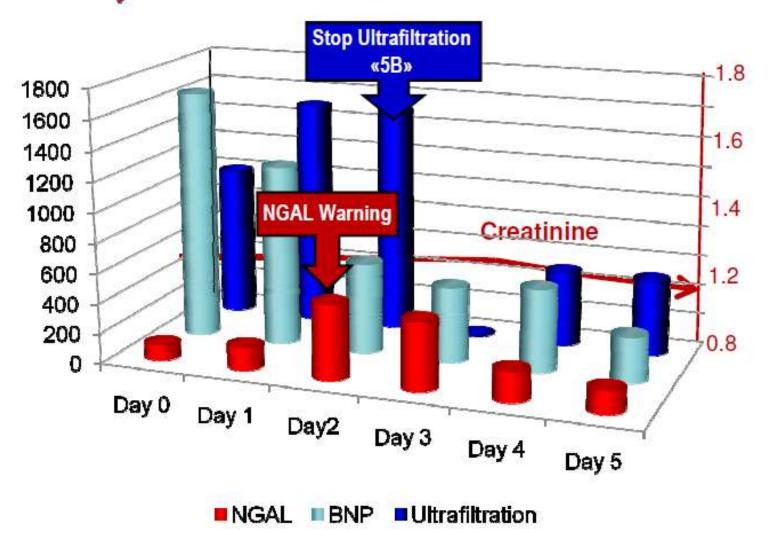




■BNP ■Ultrafiltration

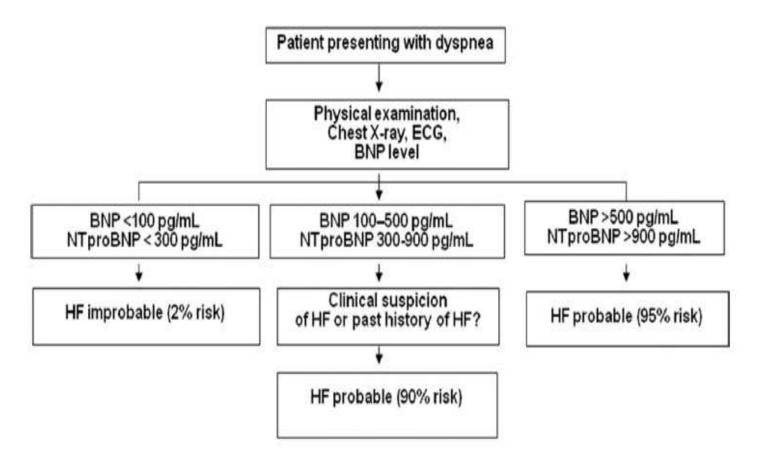


HF, Ultrafiltration and NGAL



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BNP Consensus Guidelines



Adapted from Silver MA et al. Congest Heart Fail. 2004; 10(5 suppl 3): 1–30.

Figure 6. Fluid overload leads to underestimation of severity of AKI. ECG, electrocardiogram. Reprinted with permission from Macedo et al.²⁴





BIVA





Assessment of Volume Status

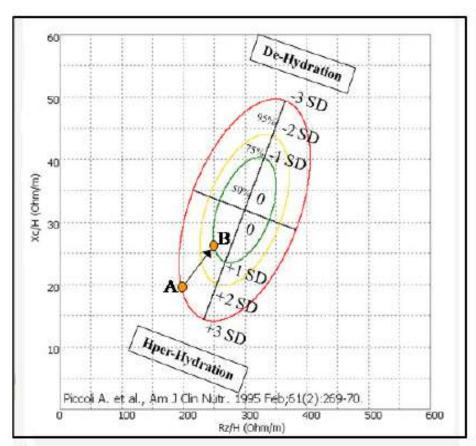
- Clinical
 - Skin Turgor
 - Capillary refill
 - Venous distention
 - Orthostasis
 - Blood Pressure
 - Organomegaly
 - Pulmonary edema
 - Urine volume
 - Urine Osmolality

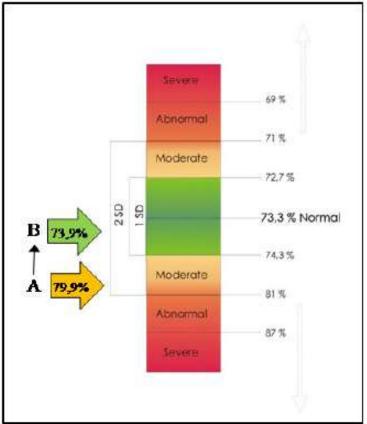
- Monitoring
 - Invasive
 - Central Venous Pressure
 - Pulmonary Artery Pressure
 - Cardiac Output (PICCO)
 - Pre-Load parameters
 - Volume responsiveness (SPV, PPV)
 - Non-Invasive
 - Echocardiography
 - Bioimpedance Spectroscopy





Optimal Hydration: How to get there?







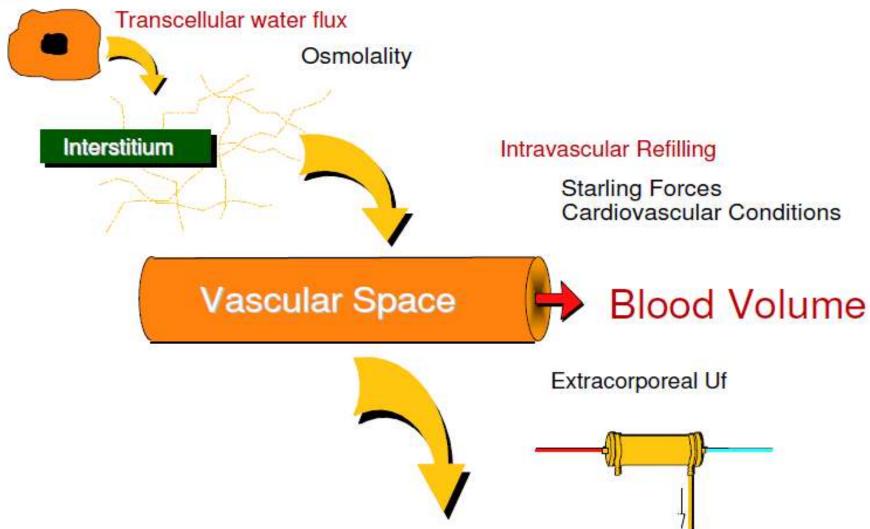


Blood Wolume



BLOOD VOLUME = Uf - Refilling









Blood Pressure

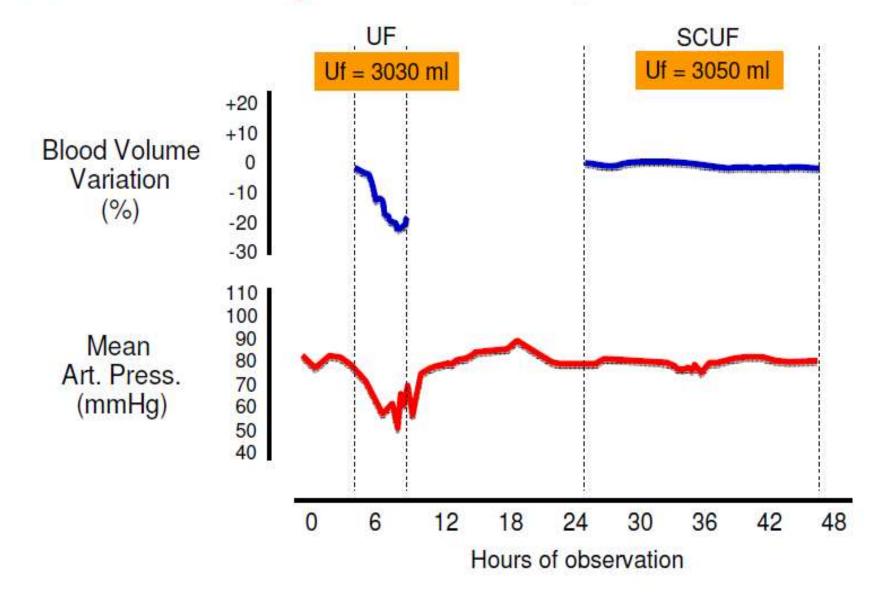
Blood pressure

- Blood pressure, as a measure of volume status, is a poor and late changing indicator.
- Orthostatic vital signs combine dynamic gravity induced changes in pulse and blood pressure that occur as a consequence of volume movement resulting from postural change.



Hemodynamic response



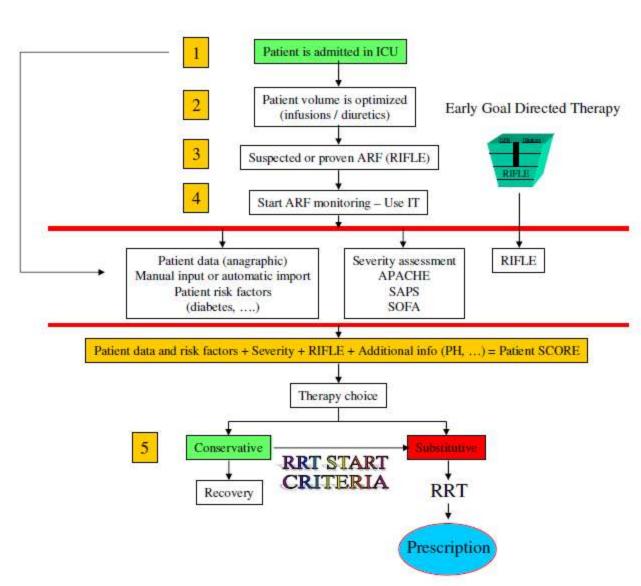


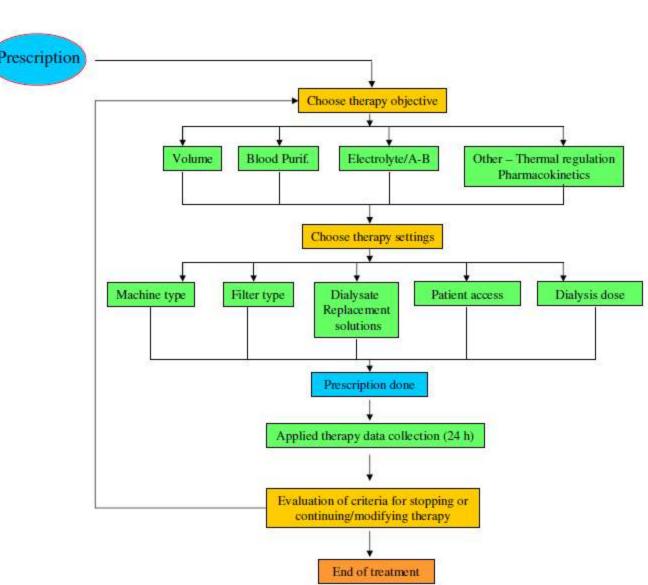
Renal problems in this lady

- AKI on top of CKD.
- What is volume status? (hypo or hyper).
- What is ideal RRT if needed?



FROM ADMISSION TO PRESCRIPTION IN FIVE STEPS





Window Help





Every patient admitted in the intensive care unit (ICU) is at high risk of developing single or multiple organ failure (MOF)

- •defects in organ perfusion
- •reduction in mean arterial pressure
- toxic effect of inflammation and sepsis.

An ongoing matter of debate is if kidneys are victims of MOF or have a primary role in causing it.



























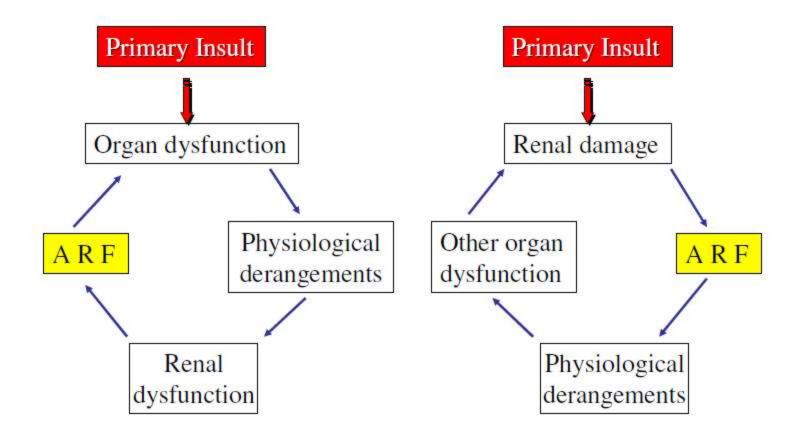








The Kidney in MOF: culprit or victim?



Help Window































Ischemic Insult

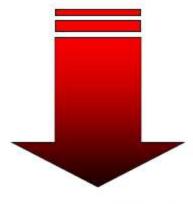
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Sepsis

Hemodynamic changes

Various **Toxins**

Pre- Renal



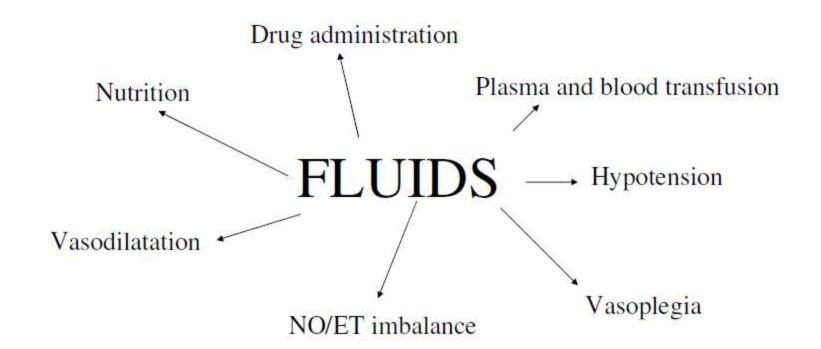
Renal cell injury







VOLUME OPTIMIZATION



Window Help



THE MOST EFFECTIVE FORM OF RENAL PROTECTION AND ARF PREVENTION



The lung: Wet you die, Dry you fly

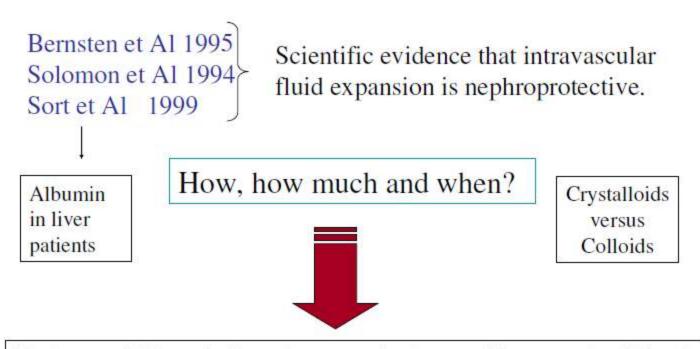
The Kidney: Dry you die, wet you pee

Window





INTRAVASCULAR VOLUME EXPANSION



No data available to indicate that a certain degree of intravascular filling is more protective to the kidney with early pre-renal dysfunction than a lesser degree of intravascular filling (e.g. RAP >15 mmHg vs 12 or 10 mmHg). Clinical judgement is still important and it should integrate data.





























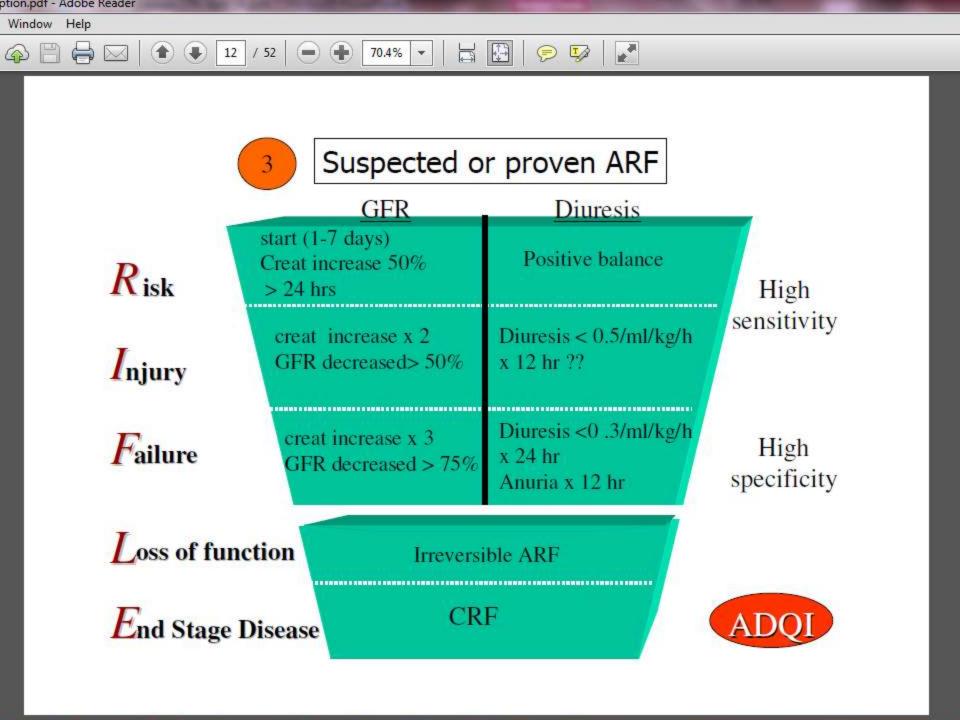


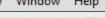
Keeping the bottles on the shelf



Watch carefully for pseudo-ARDS

- There is no scientific case for fluid administration if CI > 2.5 L/min/m² without inotropes
- If CI is high and the patient is hypotensive, they need vasopressors not fluids, irrespective of CVP/PAOP/LVEDV etc.
- Pursuing supranormal values is pointless and dangerous
- Be careful with the bleeding patient





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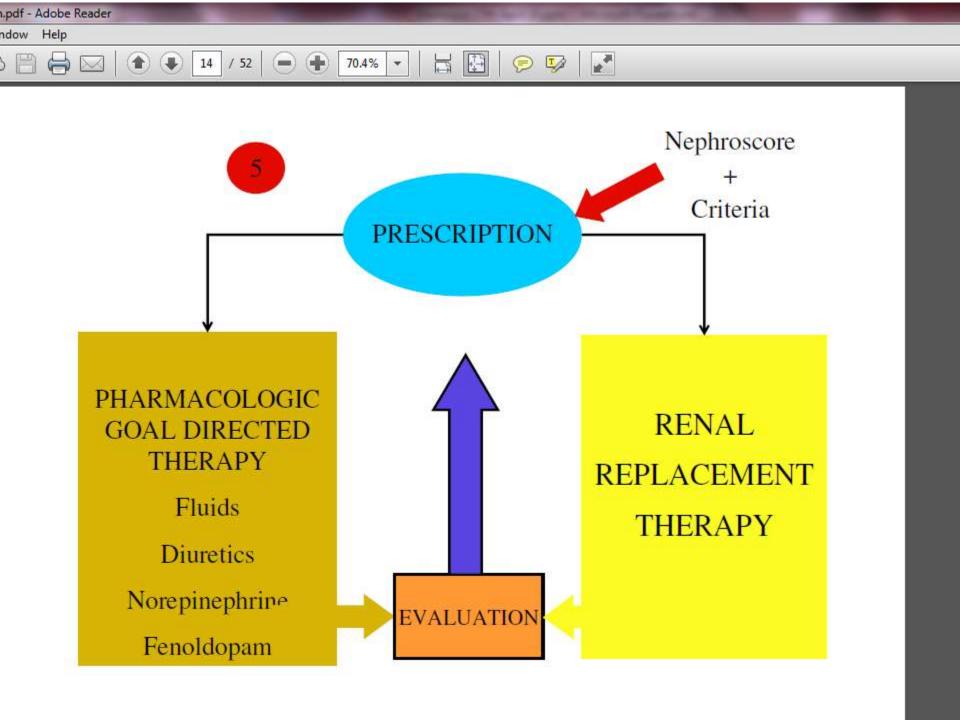


Disparate clinical data are needed and the patient must be evaluated under all aspects

Start IT ASSESSMENT

Anagraphical and clinical data Severity assessment (SAPS APACHE TISS SOFA RIFLE)

NEPHRO SCORE





























The Goal

Augmentation of perfusion pressure and oxygenation using both appropriate volume expansion and catecholamines.

The precise blood pressure targeted depends on premorbid blood pressure and individual responses but often map 90 - 100 mm hg.

The benefits of vasopressors balanced against potential adverse effects such as myocardial ischemia or tachyarrhythmia



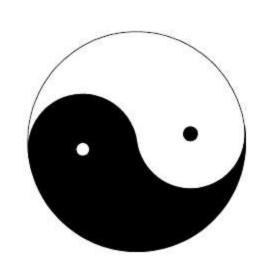
Starting treatment of A R F

How aggressive should the treatment be?

How early should the treatment start?

What should be the criteria for initiating RRT?





Treatment related risks

Do we have standard criteria and fixed reference numbers?































RRT in the ICU: STARTING CRITERIA

- Anuria Oliguria (diuresis ≤ 200 ml in 12 h)
- Severe metabolic acidosis (pH < 7.10)
- Hyperazotemia (BUN ≥ 80 mg/100 ml)
- Hyperkalemia ($K^+ \ge 6.5 \text{ mEq/L}$)
- Clinica signs of uremic toxicity
- Severe Dysnatremia (Na + \leq 115 o \geq 160 mEq/L)
- Hyperthermia
- Anasarca or severe fluid overload
- Multiple Organ Failure including renal dysfunction
- SIRS, Sepsis or Septic shock with renal dysfunction



B.E.S.T. Kidney

Window

































Prescription of Renal Replacement Therapy

Net Ultrafiltration Fluid balance

Adequacy and Dose Clearance/Modality

Acid-Base Solution Buffer

Electrolyte Dialysate/Replacement

Timing Schedule

Machine Modality

Operation Parameters **Parameters** Window Help

































VOLUME OPTIMIZATION





iew Window Help



Pre dialytic methods to establish volume status

- Hematocrit and plasma protein concentration
- Arterial Blood Pressure
- Central venous pressure
- Bioimpedance analysis and bioimpedance spectroscopy
- Central vena cava diameter
- Swan Ganz catheter and hemodynamic monitoring
- Chest X-Ray, CT Scan, COLD, PICCO.

Window

















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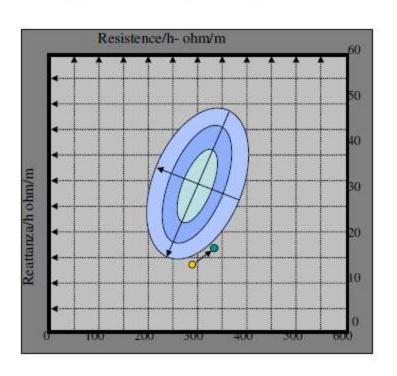




BIOIMPEDANCE

Bioimpedance can define volume status and nutrition





Early Goal Ultrafiltration Therapy

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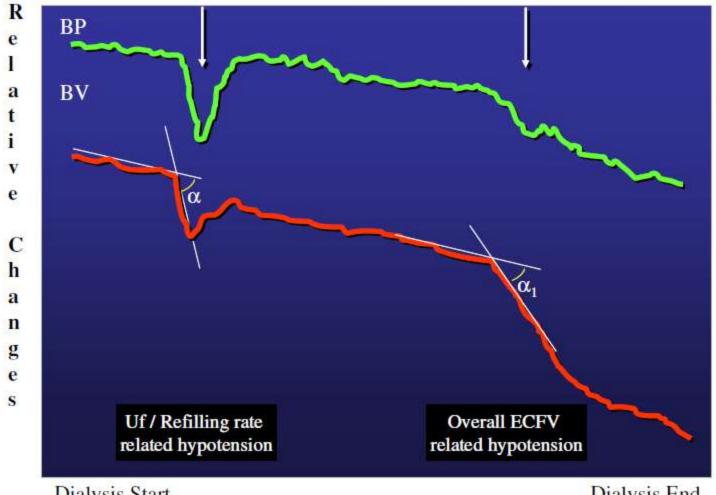






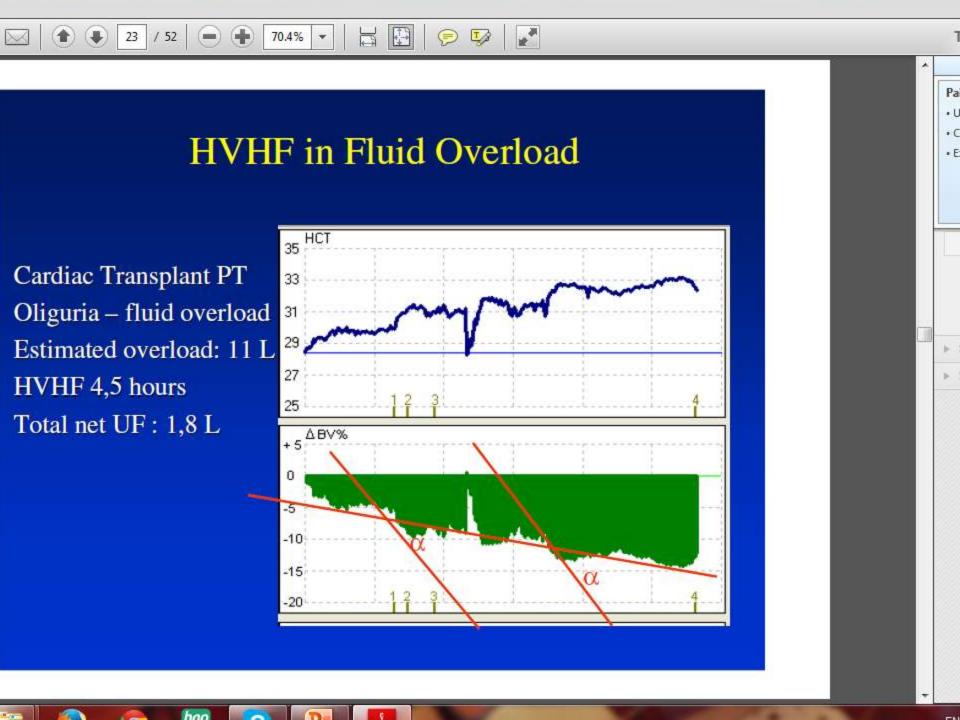


Blood Pressure/Volume Domain Map during HD and UF



Dialysis Start

Dialysis End





ADEQUACY / DOSE

Clearance: Marker Molecule – Treatment modality

Window Help























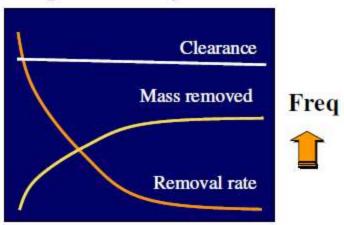




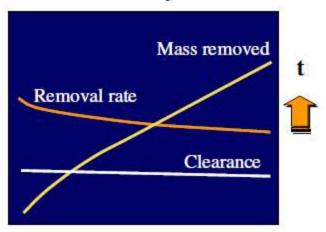




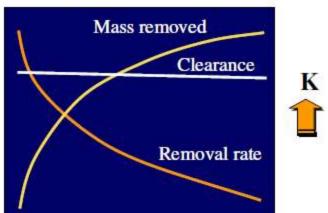
High Efficiency - Low Kc



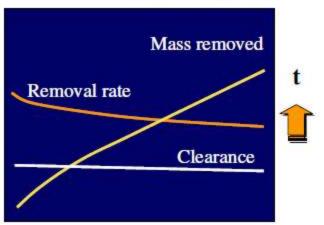
Low Efficiency - Low Kc



High Efficiency - High Kc



Low Efficiency - High Kc



































Adequate Renal Replacement in the ICU

What is treatment dose?

Treatment dose can be defined by:

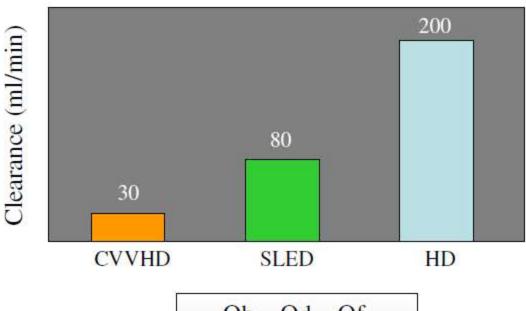
Efficiency = Inst. Clearance (K)

= Clearance x time (Kt) Intensity

Frequency = Days/week - Continuous

Efficacy = Kt/Vsp - Kt/Veq - StdKt/V

Efficiency (K) (Instant. Clearance)



K depends on:

Qb, Qd, Qf,
Reference molecule
Hemodialyzer type





























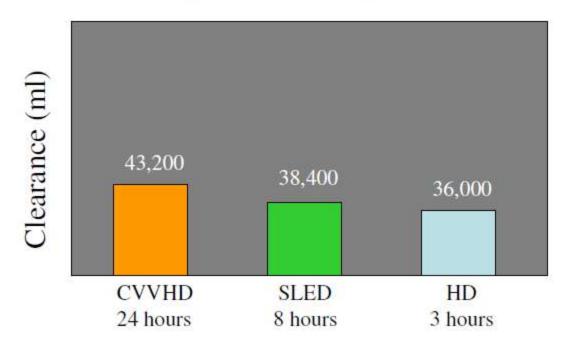




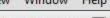


Intensity (K x t) (Daily Clearance)

 $[(ml/min) \times min)] = ml$



Window



























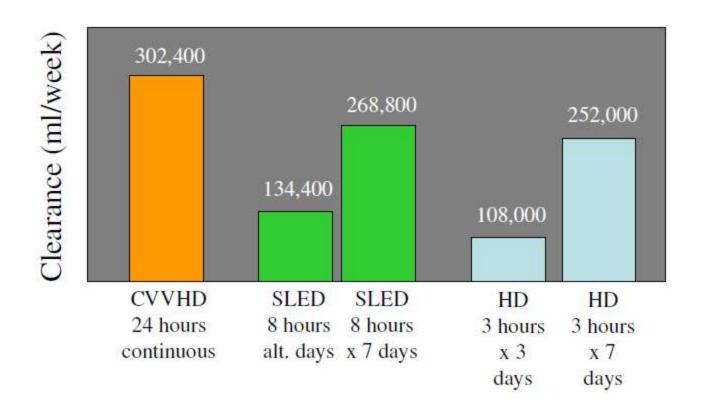






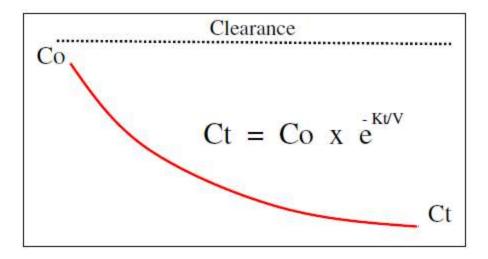
Intensity x Frequency (K x t x d/w)

(Weekly Clearance) [(ml/min) x min) x d] = ml/week





Efficacy: Fractional Clearance (K x t / V)

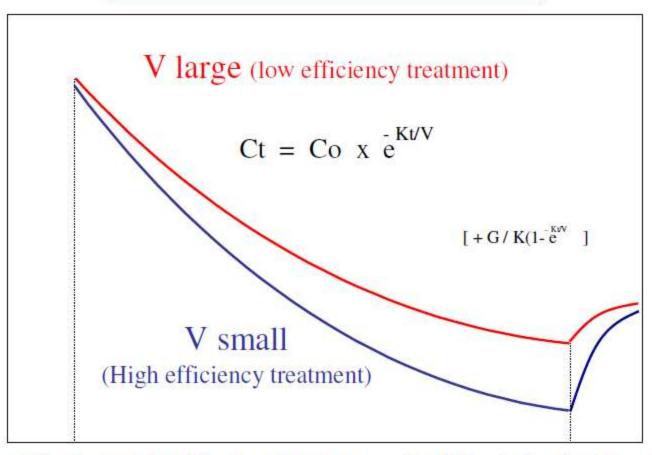


K = Average Clearance during treatment

t = Treatment time

V = Urea distribution volume (TBW)

UREA KINETIKS and VOLUME



Amount disappeared from blood > amount appeared in dialysate, i.e. blood-based vs dialysate-side kinetics overestimates urea removal. Evanson JA et Al. Kidney Int 1999; 55:1501-1506.

Vindow































QUANTITATIVE BLOOD PURIFICATION Example

D short HD

D Ext. HD

K = 200 ml/min

Urea [C]o = 110 mg/dl

Urea [C]t = 30 mg/dl

Tx time = 180 mins

Kt/V = 1.12

Tot. Clear. = 36 L

Urea removed = 18 g

Rebound = 22 %

K = 80 ml/min

Urea [C]o = 110 mg/dl

Urea [C]t = 30 mg/dl

Tx time = 480 mins

Kt/V = 1.24

Tot. Clear. = 38.4 L

Urea removed = 27 g

Rebound = 6 %

K = 30 ml/min

Urea [C]o = 70 mg/dl

Urea [C]t = 65 mg/dl

Tx time = 1440 mins

Kt/V = 0.9

Tot. Clear. = 43.2 L

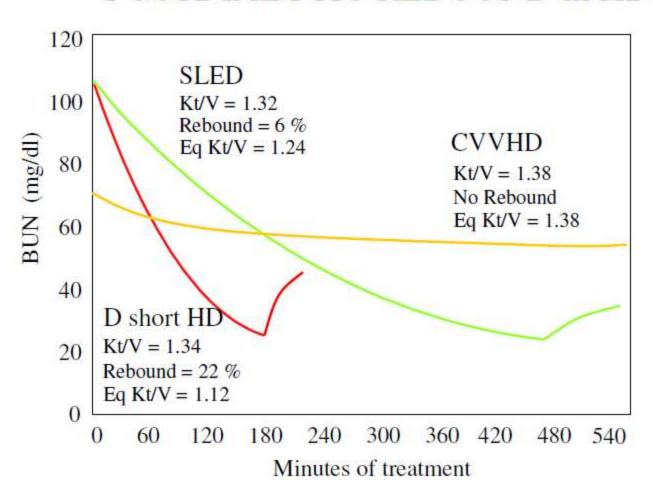
Urea removed = 33.6 g

No Rebound

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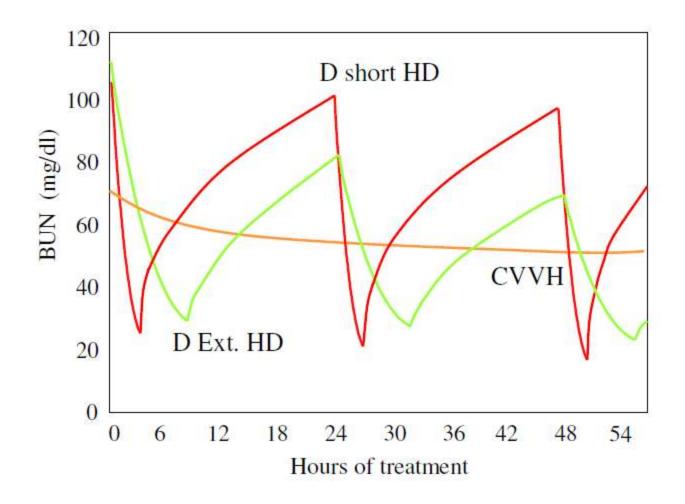


POSTDIALYTIC REBOUND in HD

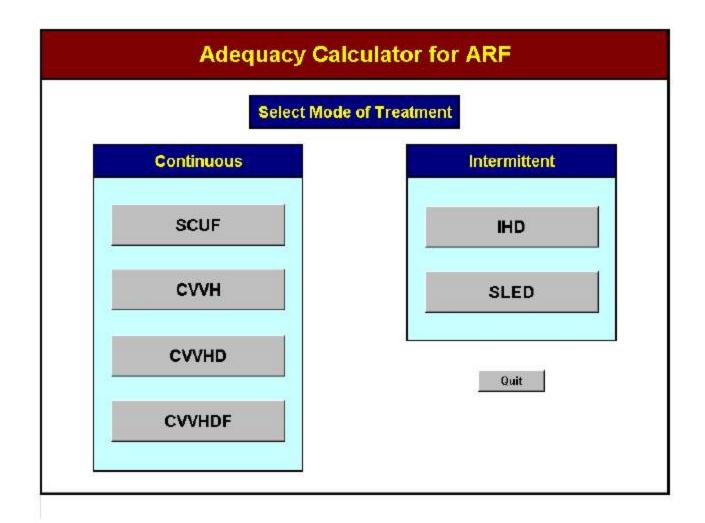


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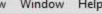








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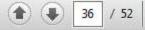






















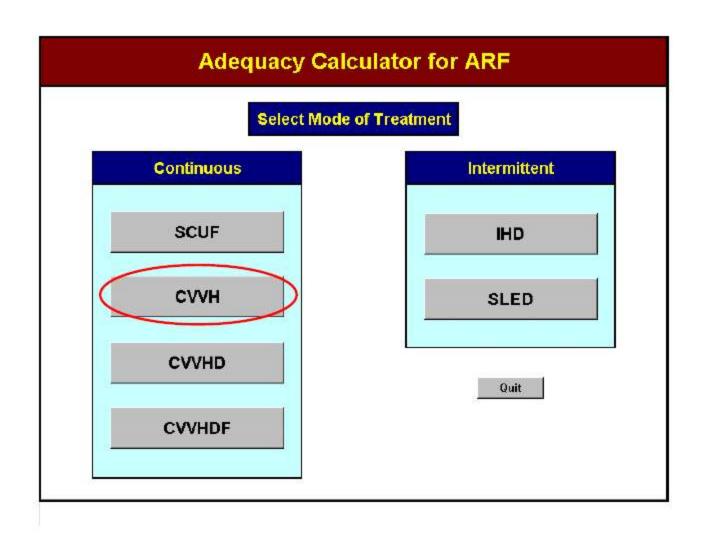










































ADEQUACY / DOSE

Clearance: Marker Molecule – Treatment modality

Fixed: Standard = 2 L/h

High Vol. = > 3 L/h

Personalized: Standard = 30-35 ml/h/Kg b.w.

High Vol. = 45 ml/h/Kg b.w.

In CVVH

ml/min

or L / 24 h

Window Help



Why Uf = Tx Dose in CVVH?

Clearance in the human kidney (K) =
$$\frac{[U] \times V}{[P]}$$

Clearance in the hemofilter (K) =
$$\frac{[Uf] \times Qf}{[P]}$$

Where: [Uf]/[P] = Sieving Coefficient (S)

Urea K =
$$\frac{[Uf] \times Uf}{[P]} = \frac{80 \times 35}{80} = 35 \text{ ml/min}$$

Constant?





Patient X.Y. = Actual Body Weight 65 Kg

Estimated Fluid Overload = 5 Kg

Early Ultrafiltration Target B.W. = 60 Kg

Estimated V = 36 Liters

48 L/24h = Kt/V: 1.3

Target is 2L/h or 33 ml/min

Placement of Adequate Vascular Access
Time = 24 h (Downtime forseen? K adjustment!)
Machine = equipment capable to perform Tx (availability?)
Blood Flow = 180 ml/min (Filtration Fraction 25%)
Replacement Solution = See Acid-base and Electrolytes

Window Help



Target is 2L/h or 33 ml/min

CVVH = ultrafiltration equals clearance (post-dil.)

CVVHD = dialysate flow equals clearance only for small molecules and for a 100% saturated effluent (depends on filter)

CVVHDF = Clearance depends on ultrafiltration, site of replacement, dialysate flow rate and its saturation.































DELIVERED AND PRESCRIBED CLEARANCE Factors affecting discrepancy

- Blood flow rate lower than that displayed by the dialysis machine Inadequate vascular access
- Dialysate/ Filtrate flow lower than that displayed by the dialysis machine. Excessive filtration fraction
- Inadequate performance of the hemofilter-hemodialyzer
 - Incorrect priming procedures
 - Loss of surface area (clotting, air)
 - Loss of permeability (clogging of the membrane)
 - High blood viscosity and hematocrit
 - Excessive filtration fraction



















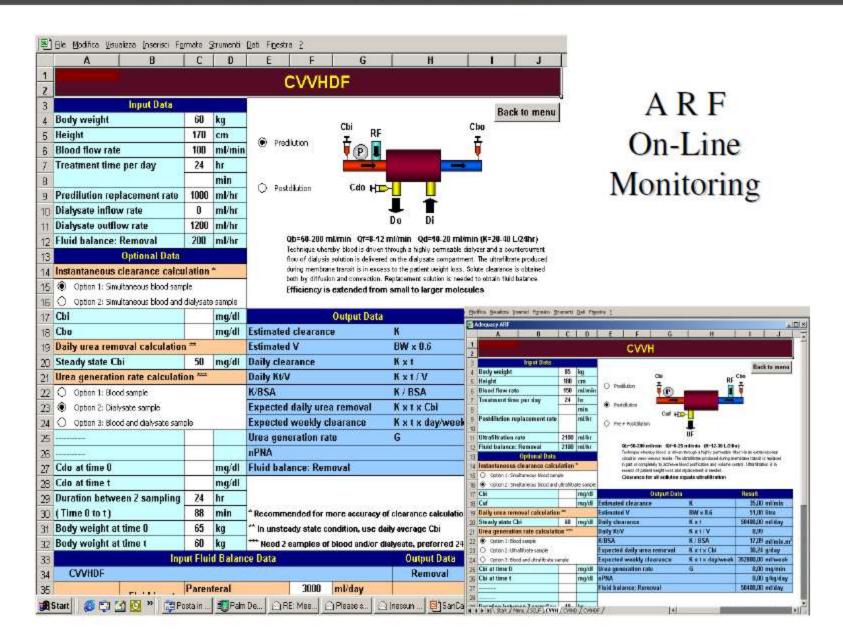












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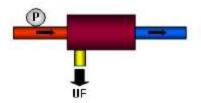






SCUF

Back to menu



Qb=50-400 ml/min Qf=2-5 ml/min

Technique where blood is driven through a highly permeable filter via an extracorporeal circuit in veno-venous mode. The ultrafiltrate produced during membrane transit is not replaced and it corresponds exactly to the patient ueight loss.

Used only for fluid control in overhydration status

| Input Fluid Balance Data | | | | | Output Data | Results |
|-----------------------------|--------------|------------------------|---------|-----------|-------------|-------------|
| SCUF | | Treatment time per day | 12 0 | hr min | Removal | 3600 ml/day |
| | | Ultrafiltration rate | 300 | ml/hr | | |
| Non CRRT | Fluid input | Parenteral | 2000 | ml/day | Repletion | 1800 ml/day |
| | | Enteral | 500 | ml/day | | |
| | Fluid output | Urine | 100 | ml/day | | |
| | | GI tract loss | 100 | ml/day | | |
| | | Insensible loss | 500 | ml/day | | |
| | | Others | 0 | ml/day | | |
| Total Fluid Balance Per Day | | | | | Removal | 1800 ml/day |





















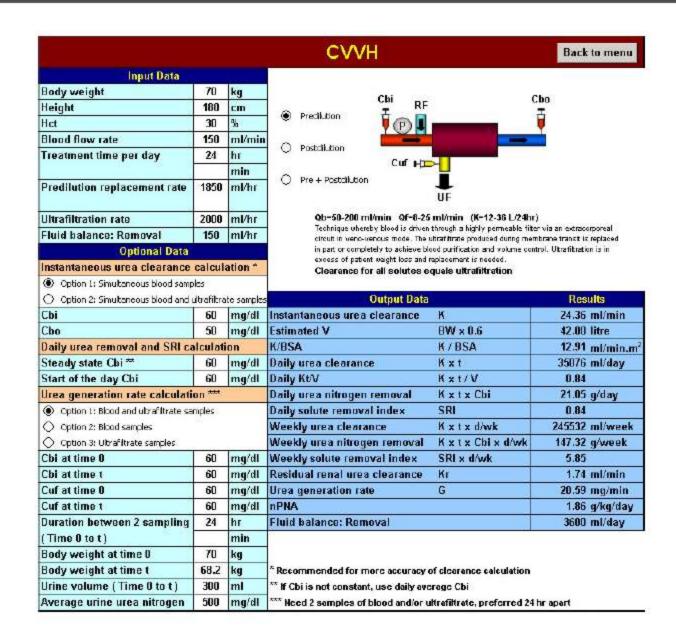






















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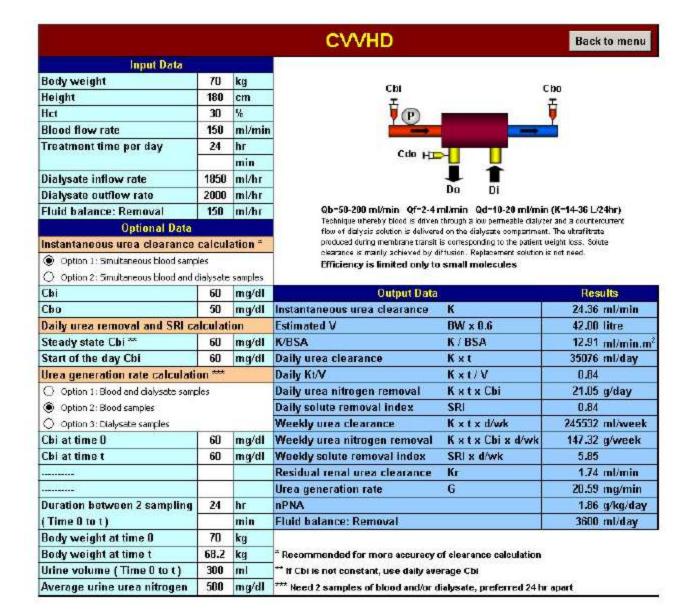












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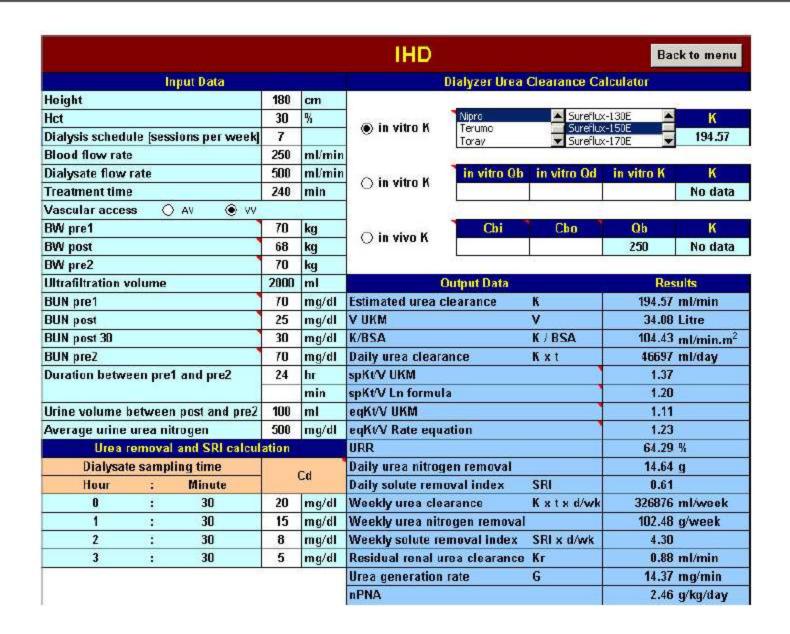






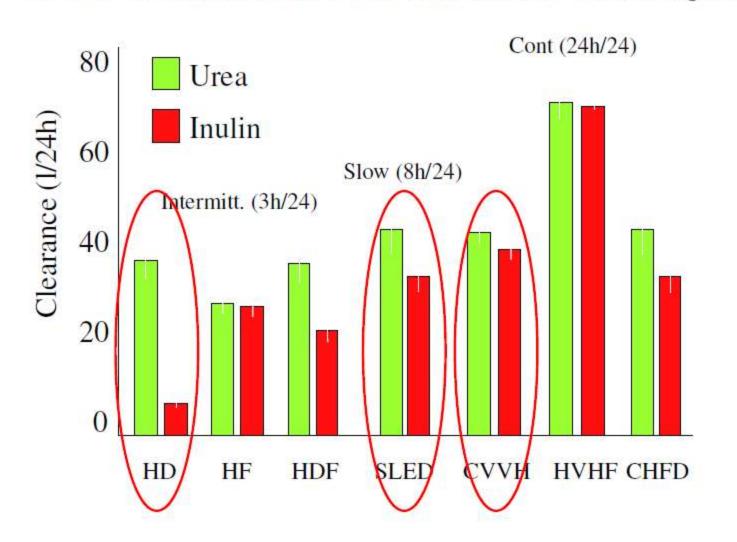


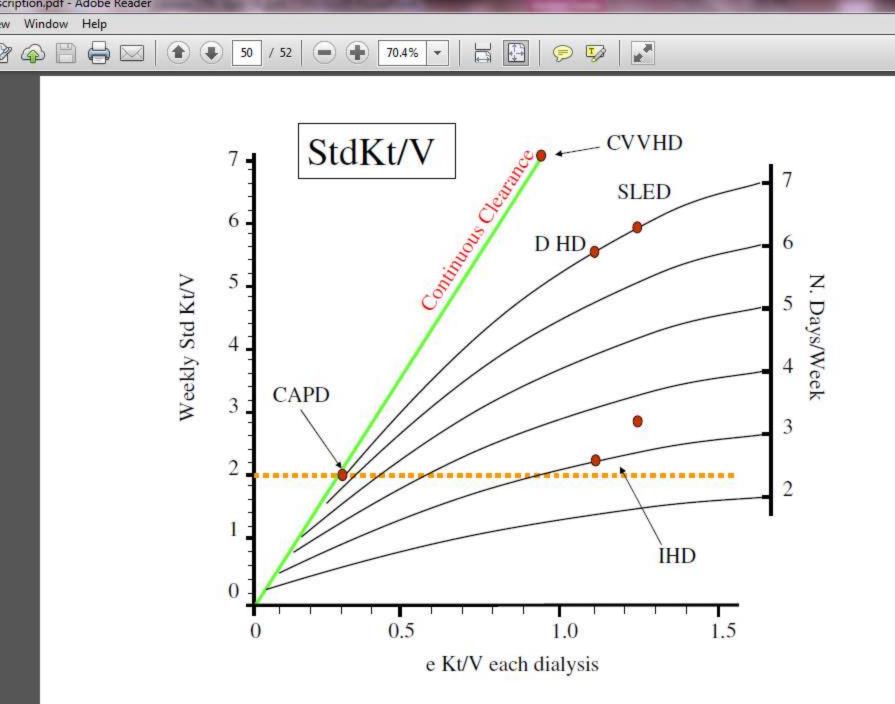




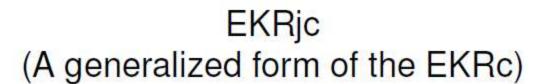
Which index of adequacy?

DAILY CLEARANCES WITH DIFFERENT TECHNIQUES









Based on core equation

51

70.4%

EKR = Continuous Removal Rate
Continuous Concentration

Sum (Σ) of net urea mass removed in each RRT over the period (i.e.weekly) Ratio from AUC of the urea timeconcⁿ profile and the duration of weekly interval (T₀-T_{wk})

EKRjc = EKRj/V x 40

Casino & Marshall, NDT 2004

Window

CONCLUSIONS

- The process from admission to therapy prescription can be standardized according to guidelines
- Prescription should be made according to patient characteristics, specific targets and available resources
- Effective delivery can be different from prescribed therapy due to several reasons including device or machine dysfunction and treatment downtime.
- Information technology can help to perform a continuous monitoring on effective delivery of therapy

Solutions for Renal problems in this lady

- CVVHDF was initiated with low volume substitution fluid replacement, net UF of 150ml/h escalating to 350ml/h. Net total UF was 8700ml.
- Plasma, packed RBCs transfusion.
- CVP started to decline.
- UOP increased reaching 100-150 ml/h.
- No orthopnea.
- Alpha methyl dopa stopped.
- Discharge from ICU.

Thank You

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